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Hierarchical Modeling of the Baroreceptor Response to Gz Acceleration and Anti-Gz Protective Equipment

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Models for Aircrew Safety Assessment: Uses, Limitations and Requirements

(la Modélisation des conditions de sécurité des équipages : applications, limitations et cahiers des charges)

Papers presented at the Specialists' Meeting of the RTO Human Factors and Medicine Panel (HFM) held at Wright-Patterson Air Force Base, Ohio, USA, 26-28 October 1998.

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Hierarchical modeling of the baroreceptor response to Gz acceleration and anti-Gz protective equipment

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1 Summary

The use of extended coverage anti-G suits and positive pressure breathing have enhanced the protection of aircrew exposed to high Gz and extreme altitudes. However, current systems do not provide optimal protection to the individual pilot during complex negative-to-positive Gz maneuvers nor do such systems adapt to changes in the physiological state of the pilot over the course of the mission, in part due to the hardware's inability to adapt to the complex response of the blood pressure regulating systems within the body. Current, medically relevant models of baroreceptor function are not suitable for the extreme changes in blood pressure and blood distribution in a body exposed to very high Gz levels of the tactical environment. Using a mix of first principle and data driven techniques, we are developing hierarchical models of baroreceptor function that include the interaction between the neuronal sub-components of the baroregulation centers of the central nervous system, the transient dynamics of pressure induced stretch in the baroreceptor organs, the effects of local pressure gradients within the aortic/carotid baroreceptor system, and the dynamic response of each of the subsystems during Gz exposures. All of these factors play a significant role in the individual's response to the Gz forces and the efficacy of the life support systems in preventing an adverse impact on cerebral blood flow and oxygenation of the brain.

2 Introduction

Environmental stresses such as acceleration, altitude, and thermal load can challenge the physiological capability of the crew of high performance tactical aircraft, such that sensory and cognitive function is degraded or even eliminated as in the extreme case of Gz induced loss of consciousness. Since World War II, experimental programs have been on-going to develop aircrew life support systems which provide an optimal degree of protection against the various stresses. However, development of computer controlled anti-G valves and breathing regulators, advances in garment technology, and the Gz stress and altitude extremes for which protection is required, makes the experimental test and validation of the systems more problematic.

Given the current environment of funding restrictions, increasing concerns regarding the short and long term hazards of acceleration and altitude exposure, and the increasing limitations imposed by ethics and human-use committees, the need for accurate models of the human response to these stresses and the efficacy of the life support systems, has become more critical. Both first principle and empirical models should be able to reduce experiment costs, minimize the exposure of test subjects to hazardous conditions, predict responses to stresses not available in experimental test facilities, and aid in predicting the short- and long-term risks associated with human experimentation as well as operational exposures.

The physiological response to high levels of negative or positive acceleration is, to a large extent, governed by the biomechanical responses of the various tissues. However, the autoregulatory mechanisms responsible for maintaining homeostasis, especially as regards blood pressure, play a critical role if the acceleration stresses last more than a few seconds. The modeling and simulation effort coordinated by DCIEM has focused on the development of rigourous 1-D finite element models of the stresses and strains in the heart during Gz exposure [6], 1-D lumped parameter models of the pressures and flows in the systemic [7] and cerebral circulations [2], along with extensive work on data driven empirical models of the physiological respones to Gz and life support equipment inflation protocols [3, 4, 5].

The body's ability to detect rapid changes in systemic blood pressure, and invoke various responses to change the capacitance and resistance of the blood vessels, as well as the rate and efficacy of the pumping heart, allows the body to adapt to a wide range of postures and exercise states. The abnormal acceleration, i.e., greater than $\pm$ 1 Gz, experienced by pilots in high performance aircraft, results in intense activation of the blood pressure control sys-

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decreased heart rate and stroke volume will in turn reduce the systemic blood pressure and the pressure in the carotid sinus.

3.3 The nerves of the carotid baroreceptor

In order to model the effect of a pressure induced strain on the properties of the baroreceptor nerve, an equivalent electrical circuit model of the nerve membrane was developed. Neuronal membranes have properties of capacitance, resistance, and voltage sources. These passive properties define how electrical impulses are transmitted along the length of the nerve. In addition to these passive properties, neuronal membranes exhibit active properties, that are voltage or chemical agent dependent, and allow the nerves to transmit information over long distances via the initiation of action potentials.

Figure 1[1] shows the equivalent electrical circuit of one finite section of the membrane of a basic barorecptor nerve. V_m represents the membrane potential. As the conducting ionic solutions inside and outside of the cell are separated by the cell membrane, the compartment acts as a capacitor, which is charged or discharged by the current flowing into or out of the cell from adjacent segments of the nerve or across the cell membrane, or by leakage currents modulated by the strain induced in the membrane.

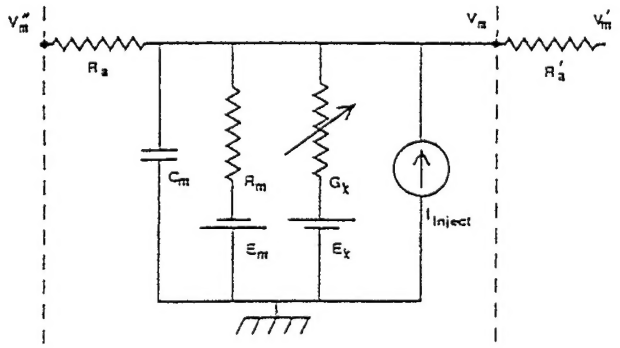

Figure 1. *Equivalent circuit model of a baroreceptor nerve*

The G_k term represents all of the various ionic conductances that give individual neurons and different neuron types their unique computational properties. Differences in the concentration of different ionic species on the inside and outside of the nerve membrane, result in a charge displacement, which in turn creates a voltage difference opposed to the flow. The fixed portion of the membrane conductance is represented by the term R_m, and is a function of the leakage current, which for most nerves is a constant.

The complexity of the model can be increased by splitting the general conductance term G_k into two components representing the flow of sodium and potassium ions. The conductances for these two predominant ions, G_K and G_{Na}, are complex functions of the membrane voltage and time, and are critical in the development of action potentials.

The deformation of the nerves embedded in the deforming arterial wall results in an increase in a strain dependent leakage current in the nerve membrane. The changes in the leakage current will in turn change the action potential firing patterns of the nerve. The highly nonlinear response of the arterial wall will be reflected in the pattern of changes in the leakage current, and thus the firing rate.

The equivalent circuit model shown in Figure 1 can be modeled with the differential equation [1]:

$$C_m \frac{dV_m}{dt} = \frac{(E_m - V_m)}{R_m} + \sum_k [(E_k - V_m)G_k] + \frac{(V_m' - V_m)}{R_a'} + \frac{(V_m'' - V_m)}{R_a} + I$$

The leakage current, I_{leak}, is in turn a complex function of the transmural pressure P across the wall of the carotid baroreceptor, i.e,

$$I_{leak} = f(P, dP/dt) \qquad (1)$$

This simple model of the baroreceptor nerve forms the basic building block to investigate a number of different physiological responses, as I_{leak} can be related to changes in the Gz environment. There are several different types of nerves in the baroreceptor, distinguished by the different firing patterns with changes in transmural pressure, as well as the speed of the action potential propogation to the brain stem neurons. The blood pressure, or more precisely, the transmural pressure across the wall of the carotid artery at the level of the carotid baroreceptor is a complex function of time, posture, and Gz forces, i.e.,

$$P = f(t, Gz(t), \alpha(t)) \qquad (2)$$

where α is the angle of the deviation of the body from the horizontal axis. In addition, P is function of external influences on the systemic circulation, such as high levels of positive pressure applied at the mouth and nose, and external counter-pressure applied to the legs and abdominal areas. Under the stress imposed by the transmural pressure gradient across the carotid arterial wall, the tissue in turn will deform in a complex frequency and time dependent manner. Models of both the deformation of the carotid tissue, and the deformation of the nerve cell membrane will have to be incorporated into the simulation.

We can extend the model by incorporating the interaction between the strain in the wall in the carotid artery and the strain in the membrane that evokes the leakage current and the effects of the viscoelastic properties of the carotid wall tissue and the neural membrane, including time-dependent relaxation and hysterisis. These may be used to explain some of the more complex firing pattern responses of individual carotid nerves.

3.4 Response to variable pressure

There are hundreds of different individual nerve endings in a human carotid baroreceptor. The threshold firing of theses nerves is pressure dependent, with various sub-populations of nerves acting in different ways to both absolute, relative, and time dependent pressure changes.

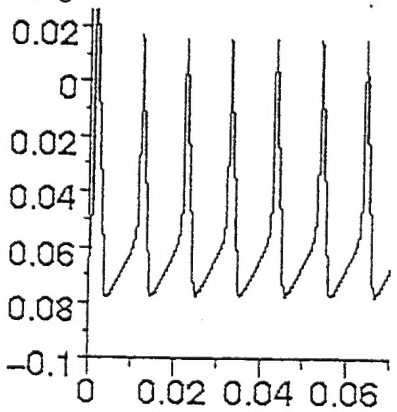

Figure 2. *Firing pattern of a carotid baroreceptor nerve at 1 Gz.*

A simple application of the equivalent circuit model can demonstrate the complexity of the Gz environment. The carotid baroreceptor extends along the carotid artery for approximately 1 cm. In a normal 1 Gz environment, even in a upright posture, the pressure gradient along the carotid baroreceptor will be less than 1 mmHg. However, at 10 Gz, as a result of the hydrostatic effects alone, and along with a change in the absolute transmural pressure, there is now a gradient of transmural pressure along carotid baroreceptor of approximately 10 mmHg. We used our model described above to examine the firing patterns of a nerve during 1 Gz and 10 Gz exposures.

Figure 2 shows the identical firing pattern of two nerves at opposite ends of the carotid baroreceptor in a 1 Gz environment. The input to the higher processing centres in the brain stem is identical.

Figure 3a shows shows the firing pattern from the nerve in the lower portion of the baroreceptor during exposure to 10 Gz. Figure 3b shows the firing pattern from a similar nerve in that portion of the carotid baroreceptor closer to the brain. The transmural pressure at this point in the carotid artery is such that the nerve has ceased to generate any action potentials, as the leakage current has dropped below a threshold.

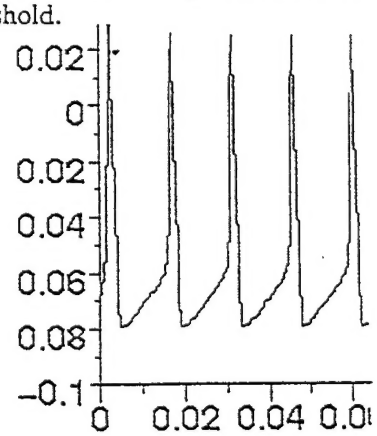

Figure 3a. *Firing pattern of a carotid baroreceptor nerve in the lower part of the carotid baroreceptor during high Gz exposure.*

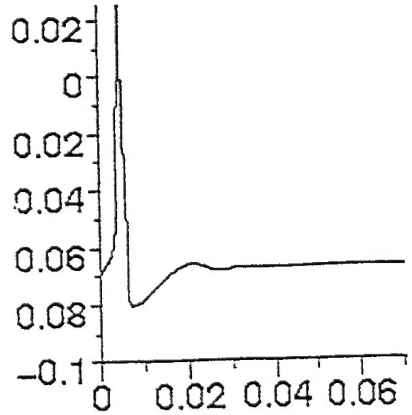

Figure 3b. *Firing pattern of a carotid baroreceptor nerve in the upper portion of the carotid baroreceptor during high Gz exposure.*

The integration of the total signal output of the carotid baroreceptor is done in the brainstem by a

number of different processing centers. A system, evolved to deal with the summed output behaviour of a wide variety of nerves responding in the 1 Gz environment, may be substantially disrupted by the effect seen in Figures 3a and 3b. Thus, the overall effector organ response calculated by the brain stem processing centers may in turn, be substantially different.

4 Discussion

In this preliminary model only a single nerve of the carotid baroreceptor sensory structure has been modeled. In the future we plan to develop a comprehensive model of the integrated response of the large population of nerves that are embedded in the carotid baroreceptor organ, the transmission of the stimulus information to the central nervous system, the integration of the sensory output of the carotid baroreceptor with other sensory input, including chemoreceptors, models of the effector nerves providing input to the peripheral blood vessels and the heart, and the response of these end organs.

An ongoing process is the integration of all of our modeling efforts into the development of a comprehensive simulation package, to provide a tool for the design and simulation of advanced aircrew life support systems.

References

[1] Bower JM, Beeman D. The Book of Genesis. TELOS 1997.

[2] Cirovic S, Walsh C, Fraser WD. A model of cerebral blood flow during sustained acceleration. In: Models for aircrew safety assessment: uses, limitations and requirements. NATO 1998, 1998; .

[3] Fraser WD, Kapps A, Askari V, Lu Z. A physiological modeling data analysis toolbox for the analysis of acceleration data. In: Models for aircrew safety assessment: uses, limitations and requirements. NATO 1998, 1998; .

[4] Fraser WD, Kapps A, Lu Z, Askari V. Modelling of the physiological respones to non-linear g-suit and positive pressure breathing schedules. In: Models for aircrew safety assessment: uses, limitations and requirements. NATO 1998, 1998;

[5] Kapps A, Fraser WD. Linear and nonlinear models of the physiological responses to negative-to-positive gz transitions. In: Models for aircrew safety assessment: uses, limitations and requirements. NATO 1998, 1998; .

[6] Tabarrok B, Behdinan K, Fraser WD. Effect of high +gz accelerations on the left ventricle. In: Models for aircrew safety assessment: uses, limitations and requirements. NATO 1998, 1998; .

[7] Walsh C, Cirovic S, Fraser WD. A model of cardiovascular performance during sustained acceleration. In: Models for aircrew safety assessment: uses, limitations and requirements. NATO 1998, 1998; .

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